Case Report Rapport de cas

Renal failure in a guinea pig (*Cavia porcellus*) following ingestion of oxalate containing plants

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Abstract — A 1-year-old guinea pig presented with anorexia, lethargy, and weight loss, 1 week after ingesting a peace lily leaf. Laboratory findings were suggestive of renal failure and included elevated blood urea nitrogen and creatinine with concurrent isosthenuria. The guinea pig was euthanized 1 month later due to worsening clinical signs.

Résumé — Insuffisance rénale chez un cobaye (*Cavia porcellus*) à la suite de l'ingestion de plantes contenant des oxalates. Un cobaye âgé d'un an a été présenté à cause d'anorexie, de léthargie et de perte de poids une semaine après avoir ingéré une feuille de *spathiphyllum*. Les résultats de laboratoire évoquaient une insuffisance rénale et comprenaient une élévation de l'azote uréique du sang et de la créatinine et une isosthénurie concomitante. Le cobaye a été euthanasié 1 mois plus tard à cause d'une détérioration des signes cliniques.

(Traduit par Docteur André Blouin)

Can Vet J 2006;47:787–789

Case description

A 1-year-old, male guinea pig (*Cavia porcellus*) was presented to the Western College of Veterinary Medicine Teaching Hospital with a 3 d history of inappetence and lethargy. The guinea pig seemed interested in food but ate only small bites or nothing at all. He also appeared to lack interest in his surroundings and was urinating and defecating less frequently than usual. The diet comprised mainly grass, lettuce, and parsley. In addition to various other fruits and vegetables, sunflower seeds and guinea pig pellets (Hagen Guinea Pig Pellets; Rolf C. Hagen, Montreal, Quebec) were provided. There was access to plain water and water to which vitamin C drops (UltraCare Vita-Sol; 8 in 1 Pet Products, Hauppauge, New York, USA) were added approximately q4d. Housing consisted of a plastic tub with metal bars on top, bedded with pine shavings. Prior history included a routine health check and nail trim 2 mo previously, during which the guinea pig was found to be healthy and in good body condition.

On physical examination, the guinea pig was quiet and listless, and his body condition was thin. He weighed 855.5 g, a decrease from his previous weight of 1227 g, 2 mo prior. Tentative diagnoses for the weight loss and lethargy included malocclusion, vitamin C deficiency, and urolithiasis. Anesthesia was mask induced and maintained on isofluorane (Isofluorane; Bimeda-MTC Animal Health, Cambridge, Ontario) while an oral examination using an otoscope, and full body radiographs were completed. Results from the oral examination were normal, and the radiographs revealed no bladder calculi or other obvious abnormalities. The guinea pig was then given vitamin C (Ascorbic Acid Injection; Univet, Milton, Ontario), 50 mg, SC. The owner was instructed to feed the guinea pig a supplement (Critical Care for Herbivores; Oxbow Pet Products, Murdock, Nebraska, USA), 50 mL/kg body weight (BW), divided into 3 to 5 feedings per day.

On day 2, the guinea pig seemed brighter and had an increased appetite. The guinea pig was again anesthetized with isofluorane and a blood sample was drawn from the cranial vena cava for a complete blood (cell) count (CBC) (Abbott Cell-Dyn 3500R; Abbott Laboratories, Chicago, Illinois, USA) and serum biochemical panel (Roche Hitachi 912; Roche Hitachi, Montreal, Quebec). An abdominal ultrasonographic examination was completed and revealed no abnormalities. The guinea pig was then placed in an empty kennel to collect a urine sample for a complete urinalysis. Another injection of vitamin C (Ascorbic Acid Injection), 50 mg, SC, was given and the owner was instructed to continue supplemental feeding while awaiting laboratory results. It was then that the owner revealed the guinea pig had ingested a leaf from a peace lily (*Spathiphyllum* spp.) 1 wk earlier and had also been chewing on the stalk of a split leaf philodendron (*Philodendron pertusum*) during the past 2 mo.

On day 3, laboratory results were obtained. The CBC revealed a mild anemia (Table 1, day 2). Abnormal serum biochemical parameters included an elevated blood urea nitrogen (BUN) and creatinine, hypercalcemia, and hyperglycemia (Table 1, day 2). The urinalysis revealed

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Table 1. The hematocrit, results from the serum biochemical panels, and the urine specific gravity (USG) on days 2 through 38

	Day 2	Day 18	Day 38	Normal range (1)	Units
Hematocrit	0.303	0.213	0.140	0.320–0.500	L/L
Glucose	12.2	9.2	9.6	3.6–6.9	mmol/L
BUN	25.7	26.6	20.6	3.2–11.2	mmol/L
Creatinine	248	315	379	53–194	μmol/L
Calcium	3.38	3.57	2.75	2.05–2.99	mmol/L
Phosphorus	1.69	2.25	2.67	0.97–2.45	mmol/L
USG	1.011	1.006	1.006	—	—

BUN — blood urea nitrogen

a specific gravity of 1.011 (Table 1, day 2); however, no crystals were seen. These laboratory findings indicated renal failure. It was recommended that the owner encourage water consumption by feeding more green vegetables and continue supplementing vitamin C by giving 50 mg, PO, q24h. The owner was also instructed to remove access to any houseplants and to monitor appetite, water consumption, urination, and defecation. Initiating SC or intraosseus fluid therapy was discussed, but the owner elected to monitor the guinea pig's progress first.

The guinea pig returned on day 18 due to decreased appetite, lethargy, and weight loss. The weight had dropped to 756.5 g. An oral examination was repeated under isofluorane anesthesia and the findings were normal. Blood was taken from the cranial vena cava for a follow-up CBC and serum biochemical panel. The results revealed that the anemia had worsened (Table 1, day 18). The hypercalcemia, azotemia, and hyperglycemia also remained (Table 1, day 18). A cystocentesis could not be completed, so the owner was instructed to collect a urine sample at home. Isotonic saline solution was administered, 25 mL, SC, in the hospital, and the owner was instructed to repeat the SC fluids q12 to 24h. The owner returned with a urine sample that was collected from a piece of plastic wrap placed on the carpet. The measured specific gravity was 1.006.

On day 30, the guinea pig was presented again; it was very lethargic and depressed. The owner was concerned because for 4 d the guinea pig had been mouthing at his food, but bringing it back out soaked with saliva, instead of swallowing it. On physical examination, the guinea pig was in thin body condition and his weight had decreased further to 685 g. An oral examination completed under isofluorane anesthesia revealed that the first 2 lower molars on each side of the mouth were growing medially into the tongue. The lower molars were trimmed using Lempert rongeurs.

On day 38, the guinea pig returned to the Teaching Hospital. The owner complained that the guinea pig was completely inappetant, producing loose feces, and seemed to shiver when held. The guinea pig now weighed 633 g and was in very thin body condition. The heart sounds were loud and a murmur could be heard. Blood was taken from the cranial vena cava under isofluorane anesthesia, and a cystocentesis was performed. Results from a CBC and serum biochemical panel included a severe anemia, azotemia, hyperphosphatemia, and hyperglycemia (Table 1, day 38). Results from the urinalysis revealed hyposthenuria, mild glucosuria, and scant calcium oxalate dihydrate crystalluria. A urine culture was negative. These findings indicated worsening chronic renal failure. On day 41, the owner requested euthanasia due to the guinea pig's unresolving inappetance, lethargy, and weight loss. The owner declined a request for a post mortem examination.

Discussion

Major differential diagnoses for anorexia, weight loss, and lethargy in guinea pigs include malocclusion, vitamin C deficiency, urolithiasis, metastatic calcification, renal disease, and toxicoses (2,3). The lack of calculi visualized on the radiographs or during ultrasonographic examination in this case was not consistent with urolithiasis. Metastatic calcification is a rare disease causing clinical signs such as muscle stiffness and renal dysfunction due to generalized soft tissue mineralization (2,4,5). The etiology has not been established; however, mineral imbalances, dehydration, and excessive dietary vitamin D have all been implicated (2). This disease is seen most commonly in older guinea pigs, so it would not be expected in this case due to the young age of the guinea pig and its adequate diet.

Guinea pigs require 15 to 25 mg of vitamin C daily. This can be achieved by adding vitamin C to drinking water at 200 to 400 mg/L (4). Supplements should be added to the water daily, as 50% of the potency is lost after 24 h in a glass or plastic container (4). Daily requirements can also be met by giving oral tablets or by feeding a good quality commercial guinea pig food that has vitamin C added to it, provided that it is fed within 90 d of the milling date (4). Adequate daily vitamin C can also be provided by feeding a quarter of an orange, 21 g of kale, or 1/3 of a kiwifriut (3). Lettuce is a poor source of vitamin C. Because supplemental drops were added to the water only q4d, vitamin C deficiency was considered in this case. However, due to the variety of fruit and vegetables in the diet, in addition to commercial guinea pig pellets, it was unlikely. Nevertheless, because many guinea pigs with concurrent diseases benefit from supplemental vitamin C, injections were administered (2).

Malocclusion causes signs of dysphagia, ptyalism, anorexia, weight loss, and eventually starvation (2). Guinea pigs have open-rooted incisors, premolars, and molars, which grow continuously; therefore, any of their teeth may overgrow. Malocclusion in guinea pigs seems to occur most commonly due to overgrowth of the most rostral cheek teeth (3). Whereas overgrown incisors may be detected on routine physical examination, premolar or molar malocclusion requires anesthesia and careful oral examination with a vaginal speculum or otoscope. Malocclusion seems to have a strong genetic predisposition; however, it is also seen with trauma, infection, and vitamin C deficiency (3). Anorexia leading to improper nutrition may have caused the overgrowth of the lower molars in this case.

Renal disease occurs most commonly in older guinea pigs. Clinical signs include depression, dehydration, weight loss, polydipsia, oliguria/polyuria, and unexpected death (3,6,7). Diagnosis is based on an elevated BUN and creatinine, nonregenerative anemia, isosthenuria, proteinuria, small kidney size, and renal biopsy, or at necropsy (6,7). Lesions of chronic renal disease include renal amyloidosis, idiopathic glomerulonephropathy, spontaneous hydronephrosis, or hypertensive nephrosclerosis (5,6). Causes of renal disease remain unclear; however, autoimmune

disease, infectious agents, chronic antigenic stimulation, and vascular disease have all been implicated (3,5,6). Acute renal failure is deemed an infrequent, but usually fatal, disease of guinea pigs (5). Suggested causes include oxalic acid poisoning following ingestion of oxalate-containing plants, such as beetroot, spinach, and rhubarb (5,8).

In this guinea pig, the development of renal failure following ingestion of oxalate-containing plants strongly suggests that they were the cause of the renal failure. Diagnosis of oxalate poisoning is based on the history of ingestion of oxalate containing plants, clinical signs, hypocalcemia, and finding calcium oxalate crystals in the urine (9). However, calcium oxalate crystals are not always seen in the urine of patients following oxalate ingestion (9). Interestingly, the hypercalcemia that was noted during the guinea pig's treatment does not coincide with the expected hypocalcemia seen in animals with oxalate poisoning. Hypercalcemia is a rare finding in guinea pigs and is usually related to hypophosphatemia from inadequate nutrition (2). Although not documented in guinea pigs, dogs occasionally exhibit mild hypercalcemia with renal failure (10). This usually occurs following conversion from oliguria to polyuria, as calcium salts that were deposited during oliguria are mobilized from soft tissues (10). Mild hypercalcemia has also been noted in some dogs with oliguric renal failure, although the mechanism has not been determined (10).

Other causes for the weight loss and inappetence in this guinea pig were ruled out, based on laboratory work, serial oral examinations, and diagnostic imaging. Specifically, urolithiasis was excluded, based on normal radiographs and ultrasonographic examination, and there was no evidence of malocclusion on repeated oral examinations. Therefore, the historical ingestion of oxalate-containing plants and the presence of renal failure based on laboratory findings, with no other cause for the acute onset of renal failure, suggested that the cause of the renal failure was the ingestion of the toxic plants. Unfortunately, a diagnosis could not be confirmed.

Peace lilies and philodendrons contain oxalates as their toxic principle and their leaves and stems are the potentially toxic parts (9,11,12). In most species, chewing on the leaves and stems results in local irritation causing oral redness and swelling, salivation, and occasionally stomach upset (9,12). However, if enough plant material containing oxalates is consumed, severe effects such as hypocalcemia and oxalate nephrosis can occur (9). Initially, oxalates are absorbed from the gastrointestinal tract and combine with serum calcium and magnesium. The sudden hypocalcemia impairs normal cell membrane function and may lead to muscle tremors, weakness, collapse, and death. Insoluble calcium oxalate crystal formation leads to irreversible kidney damage, causing eventual kidney failure. Members of the Liliaceae plant family (*Lilium* spp. and *Hemerocallis* spp.) are considered toxic to cats; however, peace lilies and philodendrons have not been documented to cause acute renal failure in cats (13). In cats, the stomatitis that usually occurs as a result of the ingestion of oxalate-containing plants likely limits the consumption of the plant, such that oxalate nephrotoxicosis does not occur.

Client education is essential to ensuring that animals do not have access to oxalate-containing plants. All guinea pig owners should be made aware of the potential renal toxicity associated with the ingestion of these plants. Examples of other houseplants that contain oxalates include Jack in the pulpit (*Arisaema triphyllum*), elephant's ear (*Alocasia* spp.), flamingo flower (*Anthurium andraeanum*), lords and ladies (*Arum maculatum*), wild calla (*Calla palustris*), caladium (*Caladium* spp.), dumb cane (*Dieffenbachia sequine*), variegated philodendron (*Epiprenum* spp.), cutleaf philodendron (*Monstera* spp.), umbrella tree (*Schefflera actinophylla*), and calla lily (*Zantedeshia aethiopica*) (9). Common garden plants that are also high in oxalates include Swiss chard, rhubarb, spinach, and beetroot. Upon acute ingestion of oxalate-containing plants, the mouth should be rinsed copiously with water (12). The animal can be given milk or another source of calcium to prevent hypocalcemia by precipitating soluble oxalates (9). Fluid therapy should be implemented to promote diuresis and aid in the removal of oxalate crystals. Oxalate nephrosis is irreversible, thus supportive care remains the only treatment option.

Acknowledgment

The author thanks Dr. Dennilyn Parker of the Western College of Veterinary Medicine for her guidance and support. CVJ

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